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	L14	L11 AND antagonist	618
	L13	L11 AND L12	65
	L12	424/130.1,141.1,142.1,143.1,145.1.CCLS.	3242
	L11	MCP-1RA OR MCP-1RB OR CCR2 OR CKR2 OR MCP-1 receptor	843
	L10	Coughlin.IN.	773
	L9	Coughlin-S.IN.	773
	L8	Coughlin-Shaun.IN.	1
	L7	Coughlin-S-R.IN.	11
	L6	Coughlin-Shaun-R.IN.	26
	L5	Charo-I-R.IN.	1
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	L2	Propagermanium OR eotaxin OR eotaxin-2 OR eotaxin-3 OR Amlodipine	2926			
	L1	(rheumatoid arthritis OR alvoclitis OR artherosclerosis)	46717			

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1. Document ID: US 6806061 B1

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L4: Entry 1 of 8

File: USPT

Oct 19, 2004

US-PAT-NO: 6806061

DOCUMENT-IDENTIFIER: US 6806061 B1

TITLE: G protein-coupled receptor gene and methods of use therefor

DATE-ISSUED: October 19, 2004

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY Gerard; Craig J. Dover MA Gerard; Norma P. Dover MA Mackay; Charles R. Newton Highlands MA Ponath; Paul D. Boston MA Post; Theodore W. Newton MA Oin; Shixin Lexington MA

US-CL-CURRENT: 435/69.1; 435/320.1, 435/325, 435/69.7, 536/23.1

Full Title Citation Front Rev	iew Classification Date	Reference	Claims KMC Draw Des

2. Document ID: US 6413967 B1

L4: Entry 2 of 8

File: USPT

Jul 2, 2002

US-PAT-NO: 6413967

DOCUMENT-IDENTIFIER: US 6413967 B1

** See image for <u>Certificate of Correction</u> **

TITLE: Inhibition of novel calcium entry pathway in electrically non-excitable cells acting as an anti-proliferative therapy

DATE-ISSUED: July 2, 2002

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY Gray; Lloyd S. Charlottesville VA Haverstick; Doris M. Charlottesville VA Densmore; John J. Charlottesville VA Szabo; Gabor Charlottesville VA

h eb bgeeef ee ef be

US-CL-CURRENT: <u>514/252.1</u>; <u>514/252.12</u>

ABSTRACT:

The present invention provides methods for screening for voltage gated (VG)-selective inhibitors, novel VG-selective inhibitors, compositions containing the same, methods for inhibiting calcium entry into electrically non-excitable cells with said VG-selective inhibitors, methods for preventing proliferation of electrically non-excitable cells with said VG-selective inhibitors as well as methods of treating autoimmune diseases, preventing graft rejections, preventing apoptosis and treating cancer with the same.

8 Claims, 17 Drawing figures Exemplary Claim Number: 1 Number of Drawing Sheets: 9

File: USPT

3. Document ID: US 6265184 B1

L4: Entry 3 of 8

Jul 24, 2001

US-PAT-NO: 6265184

DOCUMENT-IDENTIFIER: US 6265184 B1

TITLE: Polynucleotides encoding chemokine receptor 88C

DATE-ISSUED: July 24, 2001

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY

Gray; Patrick W. Seattle WA Schweickart; Vicki L. Seattle WA Raport; Carol J. Bothell WA

US-CL-CURRENT: $\frac{435}{69.1}$; $\frac{435}{252.3}$, $\frac{435}{320.1}$, $\frac{435}{325}$, $\frac{435}{471}$, $\frac{435}{71.1}$, $\frac{435}{71.2}$, $\frac{530}{350}$, $\frac{536}{23.1}$, $\frac{536}{23.5}$

ABSTRACT:

The present invention provides polynucleotides that encode the chemokine receptors 88-2B or 88C and materials and methods for the recombinant production of these two chemokine receptors. Also provided are assays utilizing the polynucleotides which facilitate the identification of ligands and modulators of the chemokine receptors. Receptor fragments, ligands, modulators, and antibodies are useful in the detection and treatment of disease states associated with the chemokine receptors such as atherosclerosis, rheumatoid arthritis, tumor growth suppression, asthma, and other inflammatory conditions.

11 Claims, 0 Drawing figures Exemplary Claim Number: 1

Full Title Citation Front	Review Classification	Date Reference	Claims	KWIC Draw Des

4. Document ID: US 5981230 A

L4: Entry 4 of 8 File: USPT Nov 9, 1999

US-PAT-NO: 5981230

DOCUMENT-IDENTIFIER: US 5981230 A

TITLE: Polynucleotide encoding chemokine .beta.-4

DATE-ISSUED: November 9, 1999

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY

Li; Haodong Gaithersburg MD Adams; Mark D. North Potomac MD

US-CL-CURRENT: <u>435/69.5</u>; <u>435/252.3</u>, <u>435/320.1</u>, <u>435/325</u>, <u>435/471</u>, <u>435/71.2</u>, <u>530/324</u>, <u>536/23.1</u>, <u>536/23.5</u>, <u>536/24.3</u>, <u>536/24.3</u>1

ABSTRACT:

Human chemokine polypeptides and DNA (RNA) encoding such chemokine polypeptides and a procedure for producing such polypeptides by recombinant techniques is disclosed. Also disclosed are methods for utilizing such chemokine polypeptides for the treatment of leukemia, tumors, chronic infections, autoimmune disease, fibrotic disorders, wound healing and psoriasis. Antagonists against such chemokine polypeptides and their use as a therapeutic to treat <u>rheumatoid arthritis</u>, autoimmune and chronic inflammatory and infective diseases, allergic reactions, prostaglandinindependent fever and bone marrow failure are also disclosed. Diagnostic assays for identifying mutations in nucleic acid sequence encoding a polypeptide of the present invention and for detecting altered levels of the polypeptide of the present invention are also disclosed.

25 Claims, 4 Drawing figures Exemplary Claim Number: 1 Number of Drawing Sheets: 3

1 Full 1	Title Citation	Front	Review	Classification	Date	Reference	Claims KMC Draw Desc

5. Document ID: US 5935568 A

L4: Entry 5 of 8 File: USPT Aug 10, 1999

US-PAT-NO: 5935568

DOCUMENT-IDENTIFIER: US 5935568 A

TITLE: Gene therapy for effector cell regulation

DATE-ISSUED: August 10, 1999

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY
Dow; Steve W. Denver CO
Elmslie; Robyn E. Denver CO
Potter; Terence A. Denver CO

h eb b g ee ef b e

US-CL-CURRENT: 424/93.21; 435/375, 435/69.1, 514/44

ABSTRACT:

The present invention provides a nucleic acid-based therapeutic composition to treat an animal with disease by controlling the activity of effector cells, including T cells, macrophages, monocytes and/or natural killer cells, in the animal. Therapeutic compositions of the present invention include superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules, depending upon the disease being treated. The present invention also relates to an adjuvant for use with nucleic acid-based vaccines. Adjuvant compositions of the present invention include an immunogen combined with superantigen-encoding nucleic acid molecules, either in the presence or absence of a cytokine-encoding nucleic acid molecule and/or chemokine-encoding nucleic acid molecules.

28 Claims, 14 Drawing figures Exemplary Claim Number: 1,3,5 Number of Drawing Sheets: 14

Full Title Citation Front Review Classification Date Reference: Claims KMC Strawistics

6. Document ID: US 5866373 A

L4: Entry 6 of 8

File: USPT

Feb 2, 1999

US-PAT-NO: 5866373

DOCUMENT-IDENTIFIER: US 5866373 A

TITLE: Polynucleotide encoding a human chemotactic protein

DATE-ISSUED: February 2, 1999

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY

Li; Haodong Gaithersburg MD Ruben; Steven M. Olney MD Sutton, III; Granger G. Columbia MD

US-CL-CURRENT: 435/69.5; 435/252.3, 435/254.11, 435/320.1, 435/325, 435/69.1, 435/91.41, 536/23.1, 536/23.5

ABSTRACT:

A human chemotactic protein polypeptide and DNA (RNA) encoding such polypeptide and a procedure for producing such polypeptide by recombinant techniques is disclosed. Also disclosed are methods for utilizing such polypeptide for preventing and/or treating for stem cell mobilization, myeloprotection and neuronal protection, to treat tumors, to promote wound healing, to combat parasitic infection and to regulate hematopoiesis. Also disclosed are antagonists against such polypeptides which may be employed to treat <u>rheumatoid arthritis</u>, lung inflammation, allergy, infectious diseases and to prevent inflammation and atherosclerosis. Diagnostic assays for identifying mutations in nucleic acid sequence encoding a polypeptide of the present invention and for detecting altered levels of the polypeptide of the present invention for detecting diseases are also disclosed.

25 Claims, 10 Drawing figures

h eb b g ee ef b e

Exemplary Claim Number: 1 Number of Drawing Sheets: 8

> Full Title: Citation Front Review Classification Date Reference Claims KMC Draw Des

7. Document ID: US 5668117 A

L4: Entry 7 of 8

File: USPT

Sep 16, 1997

US-PAT-NO: 5668117

DOCUMENT-IDENTIFIER: US 5668117 A

TITLE: Methods of treating neurological diseases and etiologically related symptomology using carbonyl trapping agents in combination with previously known medicaments

DATE-ISSUED: September 16, 1997

INVENTOR-INFORMATION:

NAME

CITY

STATE

ZIP CODE

COUNTRY

Shapiro; Howard K.

Narberth

PA

19072

US-CL-CURRENT: 514/55; 436/518, 436/74, 514/1, 514/23, 514/54, 514/811, 514/866, <u>514/878</u>, <u>514/879</u>, <u>514/903</u>, <u>514/912</u>, <u>536/1.11</u>, <u>536/20</u>

ABSTRACT:

Therapeutic compositions comprising an effective amount of at least one carbonyl trapping agent alone or in combination with a therapeutically effective of a co-agent or medicament are disclosed. The compositions are used to treat a mammal suffering from a neurological disease characterized by covalent bond crosslinking between the nerve cells, other cellular structures and their intracellular and extracellular components, with disease induced carbonyl-containing aliphatic or aromatic hydrocarbons present in mammals.

29 Claims, 0 Drawing figures Exemplary Claim Number: 1

Full Title Citation Front Review Classification Date Reference Claims KMC Draw: Desc

8. Document ID: US 5478848 A

L4: Entry 8 of 8

File: USPT

Dec 26, 1995

US-PAT-NO: 5478848

DOCUMENT-IDENTIFIER: US 5478848 A

TITLE: Inhibition of arthritis by L-type calcium channel antagonists nimodipine, nisoldipine and nifedipine

DATE-ISSUED: December 26, 1995

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY

h e b b g ee e f ef b е е

Aune; Thomas M.

Hamden

CT

US-CL-CURRENT: <u>514/356</u>; <u>514/355</u>

ABSTRACT:

The present invention comprises new methods for treating <u>rheumatoid arthritis</u>. It has been found that the L-type calcium channel antagonists are effective in treating arthritis. Nimodipine, nisoldipine, and nifedipine, are examples of specific compounds useful in the present invention.

8 Claims, 1 Drawing figures Exemplary Claim Number: 1 Number of Drawing Sheets: 1

Full Title Citation Front Review Classification Date	Reference Claims KMC Diaw.
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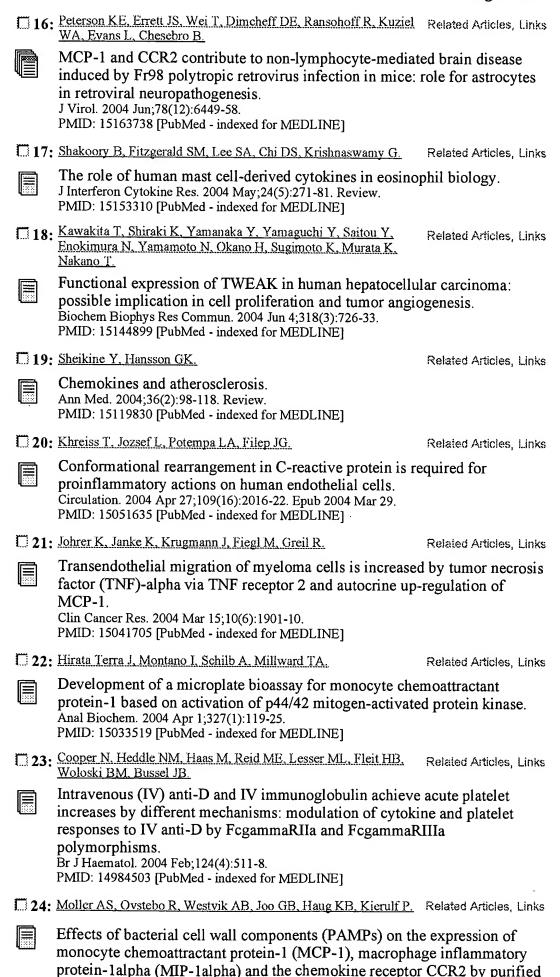
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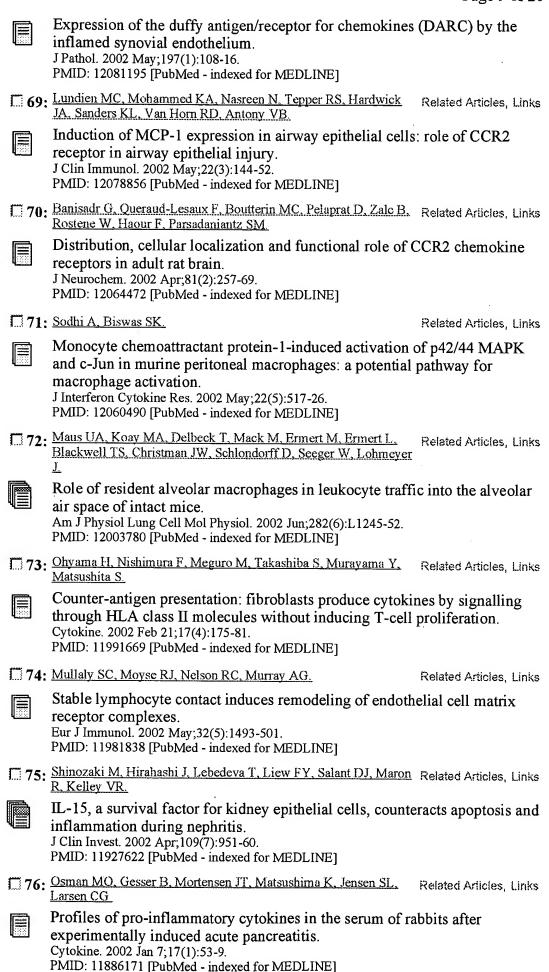


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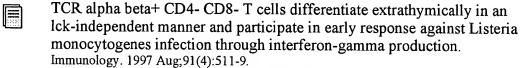
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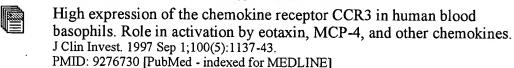
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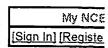
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Cytokine expression, upregulation of intercellular adhesion molecule-1, and leukocyte infiltration in experimental tubulointerstitial nephritis.

Tang WW, Feng L, Mathison JC, Wilson CB.

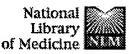
Department of Immunology, Scripps Research Institute, La Jolla, California.

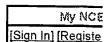
BACKGROUND: Cytokines are intercellular polypeptide messengers that mediate immune and inflammatory responses. The temporal profile of interleukin-1 beta (IL-1 beta), IL-6, tumor necrosis factor alpha (TNF-alpha), and monocyte chemotactic protein 1 (MCP-1) expression was examined in anti-tubular basement membrane (TBM) antibody-associated tubulointerstitial nephritis (TIN). EXPERIMENTAL DESIGN: TIN was induced by immunization of Brown Norway rats with bovine cortical TBM, whereas control rats received ovalbumin. Whole kidney RNA was assessed with the RNase protection assay 3, 7, 8, 9, 10, 12, and 14 days after immunization. Cytokine mRNA expression was correlated with TNF-alpha bioactivity, renal intercellular adhesion molecule-1 expression, and CD18-positive leukocyte infiltration by immunohistochemistry. RESULTS: Increased IL-1 beta, TNFalpha, and MCP-1 mRNA relative to glyceraldehyde-3-phosphate dehydrogenase appeared on day 7 when TIN involved 10 to 40% of the cortex, and peaked rapidly on day 8 when there was 60 to 80% cortical involvement (at which time 75 to 80% of the infiltrating cells were neutrophils). The increase in TNF-alpha mRNA correlated with increased bioactivity. The influx of mononuclear cells on day 8 was preceded by the expression of MCP-1 mRNA. The infiltrating leukocytes expressed the leukocyte beta 2-integrin (CD18) and were found in areas with increased intercellular adhesion molecule-1 expression. The mRNAs for IL-1 beta, TNF-alpha, and MCP-1 were undetectable by day 10 (at which time 95% of the infiltrating cells were mononuclear). An increase in IL-1 receptor antagonist mRNA paralleled those of IL-1 beta. The expression of IL-6 mRNA was similar to that for \mathbb{L} -1, except that it disappeared by day 9. CONCLUSIONS: There is a temporal association in the expression of IL-1 beta, TNF alpha, MCP-1, and IL-6 with the upregulation of intercellular adhesion molecule-1 and leukocyte infiltration within the tubulointerstitium in anti-TBM antibody-associated TIN. The narrow window of time through which these cytokines are expressed and the coincidence of their peak expression on day 8 suggest complex cytokine interactions in the pathogenesis of anti-TBM antibody TIN.

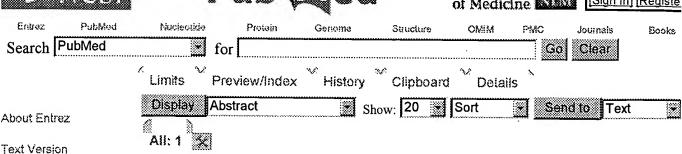
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1: J Biol Chem. 1994 Jul 1;269(26):17730-3.

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Identification and characterization of a promiscuous chemokinebinding protein in a human erythroleukemic cell line.

Horuk R, Wang ZX, Peiper SC, Hesselgesser J.

Department of Protein Chemistry, Genentech Inc., South San Francisco, California 94080.

The erythrocyte chemokine receptor is a cell surface protein that binds a wide array of chemokines including interleukin-8 (IL-8), melanoma growth stimulating activity (MGSA), monocyte chemotactic protein-1 (MCP-1), and RANTES (Regulated on Activation, Normal T Expressed and Secreted). This protein has also been identified as the Duffy blood group antigen, a cell surface receptor for the malarial parasite Plasmodium vivax. In the present study, we have identified a chemokine receptor-like binding protein in a human erythroleukemic cell line (HEL), which, based on its molecular properties, may be related to the erythrocyte chemokine receptor. Saturation binding studies with 125I-IL-8 revealed a single class of IL-8 binding sites in HEL cells with a KD of 7.4 +/- 1.9 nM and a receptor density of 12,818 +/-965 binding sites/cell. In competition studies unlabeled IL-8 MGSA, MCP-1, and RANTES were fully able to inhibit the binding of 125I-IL-8 to HEL cells. Chemical cross-linking with radiolabeled IL-8 resulted in a cross-linked species of 60 kDa in membranes from HEL cells. The labeling was specific since it was inhibited by pre-incubation with 1 microM unlabeled IL-8 or MGSA. A monoclonal antibody (Fy6) to the human erythrocyte Duffy blood group antigen/chemokine receptor blocked the binding of IL-8 and other chemokines to the HEL cell chemokine receptor-like binding protein. Cell membranes from HEL cells and from erythrocyte ghosts were subjected to SDS-PAGE and analyzed by Western blotting with anti-Fy6. The antibody bound to a molecule with a molecular mass of 50 kDa in HEL cell membranes and 40 kDa in erythrocyte ghosts. Northern blot analysis of mRNA revealed that the HEL chemokine-binding protein hybridized to a cDNA probe to the Duffy antigen/chemokine receptor.

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Differential expression of macrophage inflammatory protein-2 and monocyte chemoattractant protein-1 in experimental glomerulonephritis.

Tam FW, Karkar AM, Smith J, Yoshimura T, Steinkasserer A, Kurrle R, Langner K, Rees AJ.

Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London, England, United Kingdom.

We examined the relation between glomerular expression of chemokines from alpha-subfamily (macrophage inflammatory protein-2, MIP-2) and betasubfamily (monocyte chemoattractant protein-1, MCP-1) and infiltration of neutrophils and monocytes in antibody mediated glomerulonephritis in rats. In the accelerated model of nephrotoxic nephritis (NTN), glomerular expression of MIP-2 and MCP-1 genes correlated with the sequential migration of neutrophil and monocyte influx, respectively. These relationships were investigated further in the heterologous phase of NTN by applying various treatments known to modulate the severity of injury. Pretreatment with bacterial lipopolysaccharide resulted in greater injury, MIP-2 expression increased 25- to 50-fold, and the glomerular neutrophil count increased twoto fourfold. Both MIP-2 mRNA levels and neutrophil infiltration were reduced by additional pretreatment with IL-6, IL-1 receptor antagonist, soluble IL-1 receptor or soluble TNF receptor (Spearman correlation coefficient r = 0.897, P < 0.005). In the heterologous phase of NTN, different pre-treatments only resulted in trivial changes in MCP-1 expression and monocyte infiltration. In conclusion, glomerular MIP-2 gene expression correlates with neutrophil infiltration both temporally during the evolution of nephritis, and when glomerular injury is modified by treatment. Glomerular MCP-1 gene expression correlates with monocyte influx. The data show chemokines of alpha- and beta-subfamilies co-operative to cause selective and sequential migration of different leukocyte subsets during development of antibody mediated glomerulonephritis.

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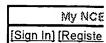
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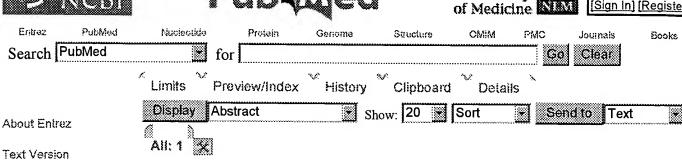
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1: J Gastroenterol. 1996 Dec;31(6):907-16.

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Alterations of the mucosal immune system in inflammatory bowel disease.

MacDermott RP.

Gastroenterology Section, Lahey Hitchcock Medical Center, Burlington, MA 01805, USA.

The normal intestinal immune system is under a balance in which proinflammatory and anti-inflammatory cells and molecules are carefully regulated to promote a normal host mucosal defense capability without destruction of intestinal tissue. Once this careful regulatory balance is disturbed, nonspecific stimulation and activation can lead to increased amounts of potent destructive immunologica and inflammatory molecules being produced and released. The concept of balance and regulation of normal mucosal immune and inflammatory events is indicative of how close the intestine is to developing severe inflammation. The normal intestinal mucosal immune system is constantly stimulated by lumenal contents and bacteria. The stimulatory molecules present in the intestinal lumen that activate and induce subsequent mucosal immunologic and inflammatory events include bacterial cell wall products, such as peptidoglycans and lipopolysaccharides, as well as other chemotactic and toxic bacterial products that are produced by the many different types of bacteria within the gastrointestinal tract. These highly stimulatory bacterial cell wall products are capable of activating macrophages and T lymphocytes to release potent proinflammatory cytokines, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-alpha). IL-1, IL-6, and TNF-alpha increase the presence of human leukocyte antigen (HLA) class II antigen-presenting molecules on the surfaces of epithelial cells, endothelial cells, macrophages, and B cells, thus increasing their ability to present lumenal antigens and bacterial products. The proinflammatory cytokines IL-1 and TNF-alpha also increase the ability of epithelial cells, endothelial cells, macrophages, and fibroblasts to secrete potent chemotactic cytokines, such as interleukin-8 (IL-8) and monocyte chemoattractant protein-1 (MCP-1), which serve to increase the movement of macrophages and granulocytes from the circulation into the inflamed mucosa. Thus, through lumenal exposure to potent, nonspecific stimulatory bacterial products, the state of activation of the intestinal immune system and mucosal inflammatory pathways are markedly up-regulated. This raises the question of whether there is a deficiency in effective down-regulation through the absence of normally suppressive cytokines such as interleukin-10 (IL-10),

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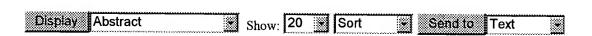
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transforming growth factor-beta (TGF-beta), interleukin-4 (IL-4), and IL-1 receptor antagonist. Normally, the turning off of the active and destructive immunologic and inflammatory events should occur following the resolution of a bacterial or viral infection that has been appropriately defended against and controlled by the mucosal immune system. In inflammatory bowel disease (IBD), however, the down-regulatory events and processes that should turn off the immunologic and inflammatory protective processes, once the pathogenic agent has been cleared, appear to be deficient or only partially effective. We may find that we ultimately are dealing with disease processes that have more than one genetic or cellular basis. The improved understanding of the immunopathophysiology of IBD will allow exploration of novel immunologic and genetic approaches, such as gene replacement therapy, administration of a suppressor cytokine or an altered cell surface antigen, the administration of humanized monoclonal antibodies directed against proinflammatory cytokines, or the development of newer strategies against fundamental cell biologic mechanisms such as adhesion molecules.

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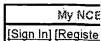
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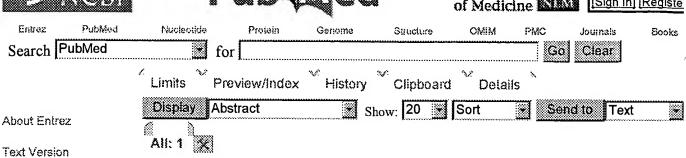
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The coordinated action of CC chemokines in the lung orchestrates allergic inflammation and airway hyperresponsiveness.

Gonzalo JA, Lloyd CM, Wen D, Albar JP, Wells TN, Proudfoot A, Martinez-A C, Dorf M, Bjerke T, Coyle AJ, Gutierrez-Ramos JC.

Millennium Pharmaceuticals, Inc., Cambridge, Massachusetts 02139, USA.

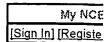
The complex pathophysiology of lung allergic inflammation and bronchial hyperresponsiveness (BHR) that characterize asthma is achieved by the regulated accumulation and activation of different leukocyte subsets in the lung. The development and maintenance of these processes correlate with the coordinated production of chemokines. Here, we have assessed the role that different chemokines play in lung allergic inflammation and BHR by blocking their activities in vivo. Our results show that blockage of each one of these chemokines reduces both lung leukocyte infiltration and BHR in a substantially different way. Thus, eotaxin neutralization reduces specifically BHR and lung eosinophilia transiently after each antigen exposure. Monocyte chemoattractant protein (MCP)-5 neutralization abolishes BHR not by affecting the accumulation of inflammatory leukocytes in the airways, but rather by altering the trafficking of the eosinophils and other leukocytes through the lung interstitium. Neutralization of RANTES (regulated upon activation, normal T cell expressed and secreted) receptor(s) with a receptor antagonist decreases significantly lymphocyte and eosinophil infiltration as well as mRNA expression of eotaxin and RANTES. In contrast, neutralization of one of the ligands for RANTES receptors, macrophage-inflammatory protein lalpha, reduces only slightly lung eosinophilia and BHR. Finally, MCP-1 neutralization diminishes drastically BHR and inflammation, and this correlates with a pronounced decrease in monocyte- and lymphocyte-derived inflammatory mediators. These results suggest that different chemokines activate different cellular and molecular pathways that in a coordinated fashion contribute to the complex pathophysiology of asthma, and that their individual blockage results in intervention at different levels of these processes.

PMID: 9653092 [PubMed - indexed for MEDLINE]









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Anti-monocyte chemoattractant protein-1 gene therapy attenuates nephritis in MRL/lpr mice.

Shimizu S, Nakashima H, Masutani K, Inoue Y, Miyake K, Akahoshi M, Tanaka Y, Egashira K, Hirakata H, Otsuka T, Harada M.

Department of Medicine and Biosystemic Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka, 812-8582, Japan.

OBJECTIVE: Monocyte chemoattractant protein-1 (MCP-1) is up-regulated and recruits and activates inflammatory cells in human diffuse proliferative lupus nephritis (DPLN) and in nephritis of lupus model MRL/lpr mice. The aim of this study was to examine whether anti-MCP-1 gene therapy inhibits the progression of nephritis in MRL/lpr mice. METHOD: An NH(2)-terminal deletion mutant of the MCP-1 gene, 7ND, was injected into skeletal muscles of MRL/lpr mice with advanced stage nephritis to blockade MCP-1 and its receptor (CCR2) signalling pathway. RESULT: Histological findings of kidneys in treated mice, which received more than four injections of 7ND, showed that protection against renal injury resulted from reduced infiltration of leucocytes. Therefore, this therapy has been shown to prolong the life span of MRL/lpr mice. CONCLUSION: Anti-MCP-1 gene therapy is specifically effective in the localized inflammatory region. The data presented here indicate that this anti-MCP-1 gene therapy may be effective adjunct in the management of DPLN.

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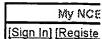
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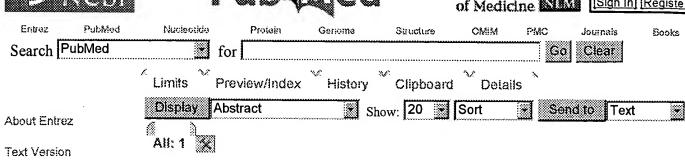
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Chemokines and atherosclerosis.

Sheikine Y, Hansson GK.

Center for Molecular Medicine, Cardiovascular Research Unit, Karolinska Institute, Stockholm, Sweden. yuri.sheikine@cmm.ki.se

Atherosclerosis is an inflammatory disease of the vessel wall, characterized by the accumulation of leukocytes, especially macrophages and T-cells. Chemokines are small heparin-binding polypeptides, whose main function is to attract cells to the areas of developing inflammation. They function by ligating G-protein coupled chemokine receptors initiating different signaling cascades. In vivo and in vitro investigations showed that chemokines are produced by a variety of cells and play important roles in the development and progression of many physiological and pathological conditions including atherosclerosis. Chemokines such as MCP-1, MCP-4, MIP-1 and RANTES may mediate leukocyte trafficking to, and their retention in, the plaque while CXCL16 seems to fulfill the dual function of a chemokine and a scavenger receptor. Chemokine and chemokine receptor homologues are secreted by several viruses, which may also play a role in the pathogenesis of atherosclerosis. Expression levels and gene polymorphisms of some chemokines may become useful clinical markers of atherosclerosis and other cardiovascular diseases. Modulation of chemokines and chemokine receptors' expression as well as their signaling pathways may provide important antiatherogenic strategies.

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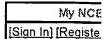
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FULL-TEXT ARTICLE

Experimental hypersensitivity pneumonitis: role of MCP-1.

Schuyler M, Gott K, Cherne A.

Department of Medicine, Albequerque Veterans Affairs Medical Center, University of New Mexico, 87108, USA.

Inhalation of Saccharopolyspora rectivirgula causes "farmer's lung" disease, a classic example of hypersensitivity pneumonitis (HP). Monocyte chemoattractant protein-1 (MCP-1) is increased in the bronchoalveolar lavage fluid of mice challenged with S rectivirgula, and S rectivirgula induces MCP-1 secretion by alveolar macrophages. We tested the hypothesis that MCP-1 and its receptor CC chemokine receptor-2 (CCR2) are essential to the development of experimental HP by treating mice with MCP-1 antibody and using CCR2(-/-) mice. Administration of anti-MCP-1 did not change the response to intratracheally administered S rectivirgula. CCR2(-/-) animals responded in a fashion similar to that of wild-type animals to intratracheally administered.S rectivirgula. To determine the influence of the MCP-1-CCR2 interaction in vitro, we transferred S rectivirgula-cultured spleen cells from S rectivirgula-sensitized mice, to naive recipients. Later, challenge of the recipients with intratracheal S rectivirgula and examination of both lung histology and bronchoalveolar lavage fluid characteristics were used to determine whether adoptive transfer had occurred. We found that cultured cells from CCR2(-/-) animals were fully capable of adoptive transfer. We conclude that interaction of MCP-1 with CCR2 is not necessary for the development of pulmonary inflammation in response to intratracheally administered S rectivirgula or cells able to adoptively transfer experimental HP.

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Targeting CCR2 or CD18 inhibits experimental in-stent restenosis in primates: inhibitory potential depends on type of injury and leukocytes targeted.

Horvath C, Welt FG, Nedelman M, Rao P, Rogers C.

Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA.

A central role for leukocytes in neointimal hyperplasia after arterial injury is suspected. However, the relative importance of neutrophils and monocytes in balloon or stent-induced injury are not well understood, and mechanistic targeting of leukocyte recruitment or function is crude. We determined the temporal and spatial distribution of different leukocytes after balloon and stent-induced injury in primate iliac arteries. Based on these data, we targeted neutrophil and monocyte recruitment selectively after angioplasty or stent implantation and demonstrated that monocyte-specific blockade achieved via blockade of the MCP-1 receptor CCR2, was effective at reducing neointimal hyperplasia after stenting. In contrast, combined neutrophil and monocyte blockade achieved by targeting the leukocyte beta(2)-integrin beta-subunit CD18 was required to reduce neointimal hyperplasia after balloon injury. Distinct patterns of leukocyte infiltration in balloon versus stent-injured arteries predict distinct mechanisms for antiinflammatory strategies targeting neutrophils or monocytes in primates and may assist design of effective clinical strategies for optimizing vascular interventions.

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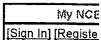
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An anti-inflammatory drug, propagermanium, may target GPI-anchored proteins associated with an MCP-1 receptor, CCR2.

Yokochi S, Hashimoto H, Ishiwata Y, Shimokawa H, Haino M, Terashima Y, Matsushima K.

Central Research Laboratory, Sanwa Kagaku Kenkyusho Co., Ltd., Hokuseicho, Inabe-gun, Mie 511-0406, Japan.

Monocyte chemoattractant protein-1 (MCP-1) promotes the migration and activation of monocytes and plays a pivotal role in the development of chronic inflammation. Propagermanium (3-oxygermylpropionic acid polymer) has been used as a therapeutic agent against chronic hepatitis B in Japan. We report here that propagermanium specifically inhibits in vitro chemotactic migration of monocytes by MCP-1. Propagermanium did not inhibit binding of MCP-1 to a human monocytic cell line, THP-1 cells, or affect intracellular Ca(2+) mobilization or the cAMP concentration in MCP-1-treated THP-1 cells. The effect of propagermanium seems to require glycosylphosphatidylinositol (GPI)-anchored proteins, as cleavage of GPI anchors by phosphatidylinositol-phospholipase C (PI-PLC) eliminated the inhibitory activity of propagermanium. Anti-GPI-anchored protein antibodies, such as anti-CD55 and anti-CD59, reduced staining of C-C chemokine receptor 2 (CCR2) with an anti-CCR2 antibody against the N-terminus of CCR2 in a flow cytometric analysis, and these antibodies also selectively inhibited MCP-1-induced migration of THP-1 cells. Furthermore, under fluorescence microscopy, GPI-anchored proteins colocalized with CCR2 on THP-1 cells. These results suggest that propagermanium may target GPIanchored proteins that are closely associated with CCR2 to selectively inhibit the MCP-1-induced chemotaxis, thus providing a mechanistic basis for the anti-inflammatory effects of the drug.

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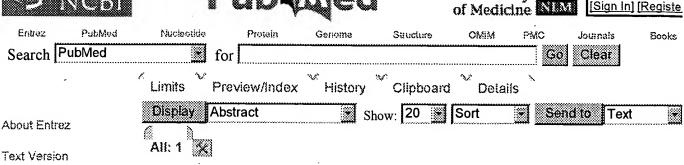
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Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

In this study, we attempted to analyze the peptide motifs recognized by 24822.111 and F9, monoclonal antibodies (mAbs) that inhibit the chemotactic activity of monocyte chemoattractant protein-1 (MCP-1), a member of the CC subfamily of chemokines. We isolated phage clones from a phage display library and identified six peptide motifs. One of these clones, C27, was strongly and specifically recognized by 24822.111 mAb, while another, G25, was similarly recognized by F9 mAb. Both the C27 motif and the G25 motif contain two cysteines in their sequences and have little homology to the primary amino acid sequence of MCP-1. These clones, however, bound to THP-1 cells, and the binding was competitively inhibited by MCP-1. The clones strongly inhibited the MCP-1-induced chemotaxis of human monocytes. The synthetic and intramolecularly disulfide-linked peptides of C27 and G25 (sC27 and sG25) also inhibited the chemotaxis induced by MCP-1, while their derivatives with serine in place of cysteine did not. suggesting the importance of the loop structure for the inhibition. These results suggest that sC27 and sG25 may mimic the MCP-1-binding domain to the MCP-1 receptor.

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Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

In this study, we attempted to analyze the peptide motifs recognized by 24822.111 and F9, monoclonal antibodies (mAbs) that inhibit the chemotactic activity of monocyte chemoattractant protein-1 (MCP-1), a member of the CC subfamily of chemokines. We isolated phage clones from a phage display library and identified six peptide motifs. One of these clones, C27, was strongly and specifically recognized by 24822.111 mAb, while another, G25, was similarly recognized by F9 mAb. Both the C27 motif and the G25 motif contain two cysteines in their sequences and have little homology to the primary amino acid sequence of MCP-1. These clones, however, bound to THP-1 cells, and the binding was competitively inhibited by MCP-1. The clones strongly inhibited the MCP-1-induced chemotaxis of human monocytes. The synthetic and intramolecularly disulfide-linked peptides of C27 and G25 (sC27 and sG25) also inhibited the chemotaxis induced by MCP-1, while their derivatives with serine in place of cysteine did not, suggesting the importance of the loop structure for the inhibition. These results suggest that sC27 and sG25 may mimic the MCP-1-binding domain to the MCP-1 receptor.

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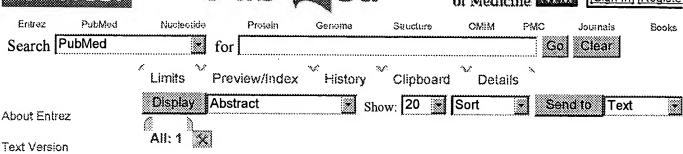
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Expression of the beta-chemokine receptors CCR2, CCR3 and CCR5 in multiple sclerosis central nervous system tissue.

Simpson J, Rezaie P, Newcombe J, Cuzner ML, Male D, Woodroofe MN.

Biomedical Research Centre and Division of Biomedical Sciences, Sheffield Hallam University, City Campus, Pond Street, South Yorkshire, S1 1WB, Sheffield, UK.

Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system (CNS) characterised by perivascular inflammatory cell infiltrates and plaques of demyelination. Chemokines have been shown to play an important role in the activation and directional migration of cells to sites of CNS inflammation. The action of chemokines requires the expression of their complementary chemokine receptors by their target cells. We have examined the expression of the beta-chemokine receptors CCR2, CCR3 and CCR5 in post-mortem MS CNS tissue using single- and double-labelling immunocytochemistry techniques. Low levels of CCR2, CCR3 and CCR5 were expressed by microglial cells throughout control CNS tissue. In chronic active MS lesions CCR2, CCR3 and CCR5 were associated with foamy macrophages and activated microglia. CCR2 and CCR5 were also present on large numbers of infiltrating lymphocytes. A smaller number of CCR3positive lymphocytes were present, but we also noted CCR3 and CCR5 on astrocytes in five of the 14 cases of MS investigated, particularly associated with processes around vessels and at the glia limitans. Ligands for CCR2 and CCR3 include MCP-1 and MCP-3 which were co-localised around vessels with the infiltrating leukocytes, but were also present in unaffected areas of cortex. The elevated expression of CCR2, CCR3 and CCR5 in the CNS in MS suggests these beta-chemokine receptors and their ligands play a role in the pathogenesis of MS.

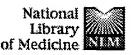
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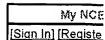
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Human endothelial cells express CCR2 and respond to MCP-1: direct role of MCP-1 in angiogenesis and tumor progression.

Salcedo R, Ponce ML, Young HA, Wasserman K, Ward JM, Kleinman HK, Oppenheim JJ, Murphy WJ.

Laboratory of Molecular Immunoregulation, Laboratory of Experimental Immunology, Division of Basic Sciences; Intramural Research Support Program, SAIC, Frederick, MD, USA.

Although several CXC chemokines have been shown to induce angiogenesis and play roles in tumor growth, to date, no member of the CC chemokine family has been reported to play a direct role in angiogenesis. Here we report that the CC chemokine, monocyte chemotactic protein 1 (MCP-1), induced chemotaxis of human endothelial cells at nanomolar concentrations. This chemotactic response was inhibited by a monoclonal antibody to MCP-1. MCP-1 also induced the formation of blood vessels in vivo as assessed by the chick chorioallantoic membrane and the matrigel plug assays. As expected, the angiogenic response induced by MCP-1 was accompanied by an inflammatory response. With the use of a rat aortic sprouting assay in the absence of leukocytic infiltrates, we ruled out the possibility that the angiogenic effect of MCP-1 depended on leukocyte products. Moreover, the direct effect of MCP-1 on angiogenesis was consistent with the expression of CCR2, the receptor for MCP-1, on endothelial cells. Assessment of supernatant from a human breast carcinoma cell line demonstrated the production of MCP-1. Treatment of immunodeficient mice bearing human breast carcinoma cells with a neutralizing antibody to MCP-1 resulted in significant increases in survival and inhibition of the growth of lung micrometastases. Taken together, our data indicate that MCP-1 can act as a direct mediator of angiogenesis. As a chemokine that is abundantly produced by some tumors, it can also directly contribute to tumor progression. Therefore, therapy employing antagonists of MCP-1 in combination with other inhibitors of angiogenesis may achieve more comprehensive inhibition of tumor growth.

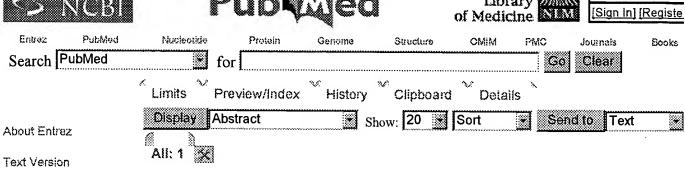
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Exaggerated hepatic injury due to acetaminophen challenge in mice lacking C-C chemokine receptor 2.

Hogaboam CM, Bone-Larson CL, Steinhauser ML, Matsukawa A, Gosling J, Boring L, Charo IF, Simpson KJ, Lukacs NW, Kunkel SL.

Department of Pathology, University of Michigan Medical School, Ann Arbor, Michigan 48109-0602, USA. hogaboam@path.med.umich.edu

Monocyte chemoattractant protein-1 is one of the major C-C chemokines that has been implicated in liver injury. The C-C chemokine receptor, CCR2, has been identified as the primary receptor that mediates monocyte chemoattractant protein-1 (MCP-1) responses in the mouse. Accordingly, the present study addressed the role of CCR2 in mice acutely challenged with acetaminophen (APAP). Mice genetically deficient in CCR2 (CCR2(-/-)) and their wild-type counterparts (CCR2(+/+)) were fasted for 10 hours before receiving an intraperitoneal injection of APAP (300 mg/kg). Liver and serum samples were removed from both groups of mice before and at 24 and 48 hours post APAP. Significantly elevated levels of MCP-1 were detected in liver samples from CCR2(+/+) and CCR2(-/-) mice at 24 hours post-APAP. Although CCR2(+/+) mice exhibited no liver injury at any time after receiving APAP, CCR2(-/-) mice exhibited marked evidence of necrotic and TUNELpositive cells in the liver, particularly at 24 hours post-APAP. Enzyme-linked immunosorbent assay analysis of liver homogenates from both groups of mice at the 24 hours time point revealed that liver tissue from CCR2(-/-) mice contained significantly greater amounts of immunoreactive IFN-gamma and TNF-alpha. The in vivo immunoneutralization of IFN-gamma or TNF-alpha significantly attenuated APAP-induced liver injury in CCR2(-/-) mice and increased hepatic IL-13 levels. Taken together, these findings demonstrate that CCR2 expression in the liver provides a hepatoprotective effect through its regulation of cytokine generation during APAP challenge.

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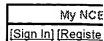
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FULL-TEXY ARTICLE

Inhibition of M-tropic HIV-1 infection by the fd phage-gene 3 protein with MIP-1alpha-binding activity.

Meta A, Torigoe N, Ito Y, Arakaki R, Nakashima H, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, 1-21-40 Korimoto, Kagoshima, Japan.

CCR5 is a chemokine receptor with seven transmembrane-domains. It is expressed on T cells and macrophages and functions as the principal coreceptor for macrophage (M)-tropic strains of HIV-1. The anti-CCR5 monoclonal antibody (mAb) 2D7 inhibits the binding and chemotaxis of the three natural beta-chemokine ligands of CCR5, macrophage inflammatory protein (MIP)-1alpha, MIP-1beta, and RANTES, to CCR5(+) cells. The mAb also efficiently blocks the infectivity of several M-tropic and dual-tropic HIV-1 strains in vitro. In this study, we attempted to determine the peptide motif recognized with the 2D7 mAb. We isolated phage clones by panning a phage display library using 2D7 and identified three peptide motifs. One of these phage clones (M23) showed a marked inhibitory activity on HIV-1 infection. The unique sequence of 15 amino acids with an internal disulfide bond was inserted in the g3p of the M23 phage clone (M23-g3p). The M23-g3p was purified by fast-performance liquid chromatography (FPLC). We show here that (1) M23-g3p was specifically recognized with anti-CCR5 mAb; (2) M23g3p showed inhibitory activity on the infectivity of M-tropic but not T-tropic HIV-1 strains; (3) M23-g3p bound to MIP-1alpha, MIP-1beta, and RANTES but not MCP-1. These results suggested that the M23-g3p might mimic the CCR5-binding domain shared by beta-chemokines, MIP-1alpha, MIP-1beta, and RANTES as well as the HIV-1 infection.

PMID: 10684964 [PubMed - indexed for MEDLINE]

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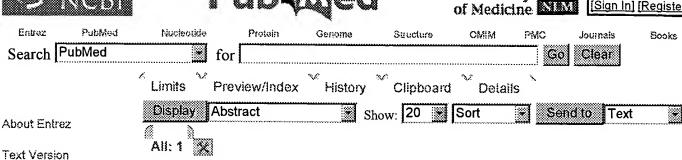
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Intracellular IL-1 receptor antagonist is elevated in human dermal fibroblasts that overexpress intracellular precursor IL-1 alpha.

Higgins GC, Wu Y, Postlethwaite AE.

Department of Pediatrics, Division of Clinical Immunology, Crippled Children's Foundation Research Center at LeBonheur Children's Medical Center, Memphis, TN 38103, USA. higginsg@pediatrics.ohiostate.edu

Cultured dermal fibroblasts from systemic sclerosis patients express higher levels of intracellular IL-1 alpha than fibroblasts from healthy controls. In this study, we found that systemic sclerosis dermal fibroblasts also express higher levels of the intracellular isoform of IL-1 receptor antagonist (icIL-1Ra) than normal fibroblasts after stimulation with IL-1 beta or TNF-alpha. A possible relationship between elevated precursor IL-1 alpha (preIL-1 alpha) and elevated ic L-1Ra was investigated by transducing normal dermal fibroblasts to overexpress preIL-1 alpha, preIL-1 beta, or icIL-1Ra. Fibroblasts that overexpressed icIL-1Ra did not have elevated levels of IL-1 alpha. On the other hand, fibroblasts that overexpressed preIL-1 alpha had at least 4-fold higher basal levels of icIL-1Ra than control fibroblasts and 4-fold higher levels of icIL-1Ra after induction with IL-1 beta or TNF-alpha. Fibroblasts overexpressing preIL-1 beta did not exhibit elevated icIL-1Ra. The differences in icIL-1Ra protein levels were reflected in differences in mRNA. In contrast, IL-1-stimulated levels of MCP-1 and IL-6 were not different in control and preIL-1 alpha-transduced fibroblasts. Addition of neutralizing anti-IL-1 alpha Abs to fibroblast cultures did not diminish basal or stimulated levels of icIL-1Ra in the preIL-1 alpha-transduced cells, supporting an intracellular site of action of preIL-1 alpha. This is the first report of an association between intracellular levels of these IL-1 family members. We hypothesize that intracellular preIL-1 alpha participates in the regulation of icIL-1Ra.

PMID: 10490999 [PubMed - indexed for MEDLINE]

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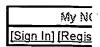
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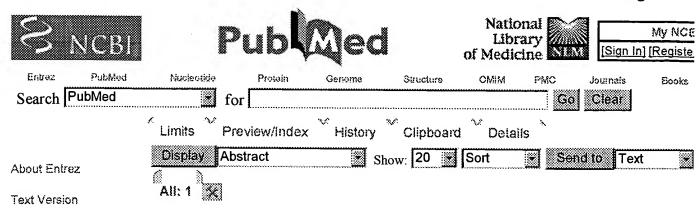
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Amlodipine, a new 1,4-dihydropyridine calcium antagonist with

a particularly strong antihypertensive profile.

Fleckenstein A, Frey M, Zorn J, Fleckenstein-Grun G.

Physiological Institute, University of Freiburg, Federal Republic of Germany.

The effects of a new 1,4-dihydropyridine derivative amlodipine have been compared with results from our previous work. Application of amlodipine at a concentration of 1.6 X 10(-6) M to isolated guinea-pig papillary muscle for 120 minutes produced a 50% reduction in tension development compared with a concentration of 3.7 X 10(-7) M nifedipine needed to produce the same result under identical conditions. This suggests that amlodipine has even weaker negative inotropic effects than nifedipine. In isolated porcine coronary strips, the K+-induced contractions were approximately 10,000 times more sensitive to the relaxing effects of nisoldipine, nitrendipine and nicardipine than to those of papaverine, whereas nifedipine and amlodipine were 3,000 times more potent than papaverine. However, in comparison with these in vitro actions, the efficacy of amlodipine appears to be greater in vivo: Simultaneous subcutaneous injection of nifedipine (20 mg/kg) and of equimolar doses of nisoldipine and felodipine attenuated the myocardial calcium uptake by rat hearts in situ (stimulated with a single subcutaneous dose of 30 mg/kg isoproterenol) with the same efficacy, whereas the actions of nitrendipine and nimodipine were considerably weaker. In contrast, amlodipine antagonized isoproterenol-stimulated myocardial calcium accumulation more effectively. Furthermore, amlodipine exhibited a high antihypertensive potency combined with rapid onset and long duration of action: Amlodipine (10 mg/kg orally [p.o.]) reduced the blood pressure of spontaneously hypertensive rats almost to the same extent as nifedipine, nitrendipine, verapamil and felodipine administered at the much higher doses of 100 mg/kg p.o. Amlodipine (20 mg/kg/day p.o.) maintained normal blood pressure during the whole life span of Dahl-S rats (5 months), but this dose is considerably lower than that reported for other 1,4-dihydropyridines. The survival of NaCl-loaded Dahl-S rats increased from 20 to 100% after administration of amlodipine (20 mg/kg/day p.o.) over 10 weeks: The effective dose of other calcium antagonists is approximately 5 times higher, but well tolerated as, e.g., demonstrated in long-term studies on Dahl-S rats with nitrendipine over 12 months. Increases in systemic arteriolar tone can be visualized in the ocular fundus of spontaneously hypertensive rats. After amlodipine (10 mg/kg p.o.) arteriolar spasm declines. Prophylaxis with 2

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doses of 20 mg/kg amlodipine daily in NaCl-loaded Dahl-S rats abolished the macroscopic and histologic changes that are normally seen in branches of the mesenteric artery. With use of electron microscopy, calcium accumulation in the lamina elastica interna was demonstrated by the potassium-pyr-oantimonate technique.(ABSTRACT TRUNCATED AT 400 WORDS)

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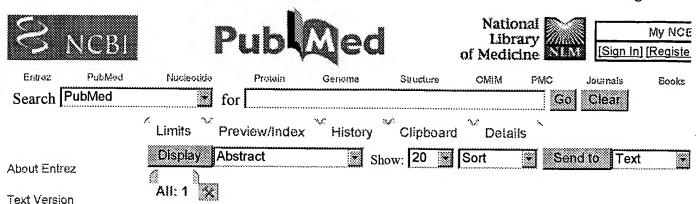
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Progress in cardioprotection: the role of calcium antagonists.

Kloner RA, Przyklenk K.

Heart Institute, Hospital of the Good Samaritan, Los Angeles, California 90017.

Calcium antagonists are now widely used for the treatment of clinical hypertension and angina pectoris. They are efficacious for the treatment of vasospastic, fixed atherosclerotic and mixed angina; they reduce the incidence of silent ischemia; and they have been shown to reduce postmyocardial infarct angina. Experimental data suggest that they may have certain cardioprotective properties in cases of acute myocardial ischemia and infarction, stunned myocardium, diastolic dysfunction, left ventricular hypertrophy and atherosclerosis. Moreover, they have been shown to improve exercise performance, as well as the diastolic abnormalities in patients with hypertrophic cardiomyopathy. In animals, they may delay or reduce the extent of myocardial necrosis after coronary occlusion or coronary occlusion followed by reperfusion, and in low doses that do not alter the hemodynamic profile, they have been shown to enhance the return of ventricular function in animals with stunned myocardium. However, the early first-generation calcium antagonists (nifedipine, verapamil, diltiazem) have not been shown to reduce myocardial infarct size or to enhance survival in patients with acute myocardial infarction. There now are clinical studies that suggest that, unlike beta blockers or nitrates, nifedipine may slow the development of atherosclerotic progression in humans over a 2-year period, and it seems likely that in the 1990s there will be further expansion of the use of calcium antagonists for not only angina and hypertension but also for aspects of cardioprotection. That calcium antagonists may delay, prevent or possibly regress atherosclerotic lesions is an exciting possibility.

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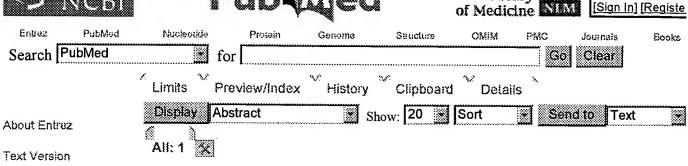
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Vascular and myocardial effects of amlodipine: an overview.

Nayler WG, Gu XH.

Department of Medicine, University of Melbourne, Austin Hospital, Heidelberg, Victoria, Australia.

Amlodipine is a long-acting dihydropyridine calcium antagonist with vascular selectivity. Although structurally related to nifedipine, amlodipine differs in several important respects, including its slow rate of onset and slow recovery. These effects probably reflect the relatively slow rate of association and dissociation of amlodipine with its binding site. The interaction of amlodipine with the calcium antagonist binding site associated with the slow Ca2+ channels differs from that of other dihydropyridines in that it involves the binding domains for the phenylalkylamine- and benzothiazepine-based antagonists, as well as for the dihydropyridines. The prolonged duration of action of amlodipine makes it suitable for use in conditions where calcium channel blockade is required on a 24-h basis. To determine whether amlodipine has a vascular protective effect, amlodipine was given orally to either cholesterol-fed rabbits or stroke-prone hypertensive rats. In the cholesterol-fed rabbits amlodipine (1 or 5 mg/kg/day) produced a significant, dose-dependent reduction in the incidence of atheromatous lesions in the thoracic aorta over an 8-week period. In stroke-prone rats the administration of amlodipine at a dose of 5 mg/kg/day reduced the incidence of mortality over a 30-week treatment period. In spontaneously hypertensive rats amlodipine (5 mg/kg/day) caused a fall in systolic blood pressure, accompanied by a significant (P less than 0.01) reduction in cardiac hypertrophy. When administered intravenously (0.25 mg/kg) 5 h before hearts were excised and made globally ischaemic for short periods (the 'stunned' heart) amlodipine pretreatment improved functional recovery associated with reperfusion.

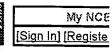
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Protective effects of various calcium antagonists against experimental arteriosclerosis.

Fleckenstein-Grun G, Frey M, Thimm F, Fleckenstein A.

Study Group for Calcium Antagonism, University of Freiburg, Germany.

Arterial walls altered by sclerotic processes accumulate lipids (particularly cholesterol) and calcium. Whereas the accumulation of lipids has long been incriminated as the major pathogenic factor involved in arteriosclerosis, concomitant arterial calcium overload has been considered of secondary importance. Using various animal models and specific calcium antagonists as experimental tools, we have shown the crucial role of excessive calcium uptake into arterial walls in the pathogenesis of arteriosclerotic lesions. Anticalcinotic vasoprotection with calcium antagonists has been demonstrated using light and electron microscopy, radiocalcium uptake experiments and calcium analyses with atomic absorption spectroscopy. The new 1.4dihydropyridine calcium antagonist amlodipine has been shown to inhibit calcium accumulation in the internal elastic membrane of abdominal arteries of NaCl-loaded salt-sensitive Dahl-S rats, and consequently also exerts protective effects against arteriosclerotic lesions, shown particularly in the distal mesenteric artery branches. Formation of human coronary plaques is marked by a substantial local uptake of calcium, whereas there is a large overlap in the mural cholesterol content of healthy coronary arteries and plaques. Experimental findings in animals and with human tissue indicate that calcium antagonists such as amlodipine may provide a new approach to the prophylaxis of coronary artery lesions.

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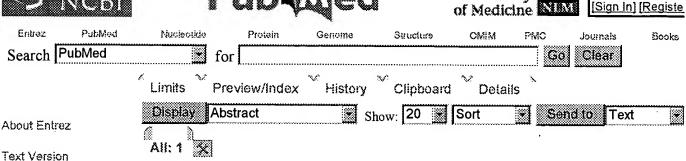
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Characterization of the CCR2 chemokine receptor: functional CCR2 receptor expression in B cells.

Frade JM, Mellado M, del Real G, Gutierrez-Ramos JC, Lind P, Martinez-A C.

Department of Immunology and Oncology, Centro Nacional de Biotecnologia, Consejo Superior de Investigaciones Cientificas, Campus Cantoblanco, Universidad Autonoma, Madrid, Spain.

We have derived anti-human CCR2-specific mAbs by immunization with synthetic peptides corresponding to CCR2 sequences presumably involved in the interaction with its ligand(s). The characterization of these mAbs includes the ability to recognize the CCR2 receptor specifically, as well as the function based on their ability to promote Ca2+ influx or to block MCP-1-induced Ca2+ influx and chemotaxis. One mAb (MCP-1 R02) that is directed to the NH2 terminal domain of the CCR2 receptor has MCP-1 agonist activity, and two that recognize the third extracellular domain (MCP-1R04 and MCP-1 R05) have MCP-1 antagonist activity. We analyzed the presence of CCR2 in several PBL and tonsil-derived leukocyte populations and found expression of this receptor in monocytes, activated T cells, and, surprisingly, in B cells. CCR2 receptor expression in B cells was further corroborated in Southern blot using CCR2-specific probes. Moreover, both MCP-1 and the agonist mAb trigger specific B cell migration via a PTX-sensitive mechanism, indicating the presence of a functional CCR2 receptor in these cells.

PMID: 9548499 [PubMed - indexed for MEDLINE]

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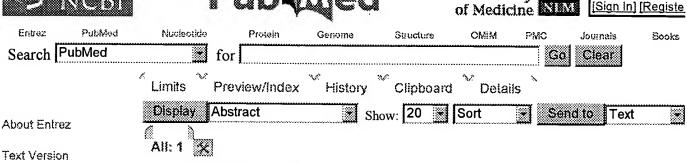
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Eotaxin is a natural antagonist for CCR2 and an agonist for CCR5.

Ogilvie P, Bardi G, Clark-Lewis I, Baggiolini M, Uguccioni M.

Institute for Research in Biomedicine, Bellinzona, Switzerland.

Eotaxin is a potent inducer of eosinophil chemotaxis and was considered as a selective ligand of the CC chemokine receptor 3 (CCR3), which is expressed on eosinophils, basophils, and Th2 lymphocytes. This study shows that eotaxin also interacts with CCR2 and CCR5 and can, thus, affect the responses of monocytes, which express both receptors. In human monocytes pretreatment with eotaxin decreased responsiveness to MCP-1, a selective ligand for CCR2, as well as to RANTES and MIP-1 beta, which bind to CCR5. Similar effects were obtained with transfected cells expressing CCR2 or CCR5, but here a difference became apparent: Eotaxin triggered CCR5 at a concentration of 100 nM but not CCR2 even at 1 microM, suggesting an antagonistic effect on this receptor. In agreement with this observation, eotaxin induced internalization of CCR5 but not of CCR2 in human monocytes and transfected cells. Binding studies showed that eotaxin displaces (125) I-MCP-1 from monocytes in a concentration-dependent manner, and functional experiments showed that eotaxin inhibits MCP-1induced chemotaxis and enzyme release. The results demonstrate that eotaxin is a CCR5 agonist and a CCR2 antagonist. The present findings suggest a role of eotaxin in the fine-tuning of cellular responses occurring at sites of allergic inflammation, in which both MCP-1 and eotaxin are produced. (Blood. 2001;97:1920-1924)

PMID: 11264152 [PubMed - indexed for MEDLINE]

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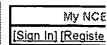
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Eotaxin is a natural antagonist for CCR2 and an agonist for CCR5.

Ogilvie P, Bardi G, Clark-Lewis I, Baggiolini M, Uguccioni M.

Institute for Research in Biomedicine, Bellinzona, Switzerland.

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PMID: 11264152 [PubMed - indexed for MEDLINE]

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The generation and characterisation of antagonist RNA aptamers to MCP-1.

Rhodes A, Smithers N, Chapman T, Parsons S, Rees S.

Molecular Discovery Department, Glaxo Wellcome Research and Development, Stevenage, Herts SG1 2NY, UK. adr7003@gsk.com

Monocyte chemoattractant protein-1 (MCP-1) has been implicated as a powerful pro-inflammatory mediator and may represent a potentially important, therapeutic opportunity for treatment of inflammatory disease and atherosclerosis. To further investigate the role of MCP-1 in inflammatory disorders we have isolated a series of RNA aptamers that bind specifically to mouse MCP-1. The highest affinity aptamers, designated ADR7 and ADR22, have been functionally characterised in vitro and in cell based assays. ADR7 and ADR22 have an affinity of 180 pM and 370 pM respectively for mouse MCP-1, they can antagonise MCP-1 binding to heparin and specifically antagonise MCP-1 induced chemotaxis in a cell based assay. An interesting feature of ADR22 but not ADR7 is that it is capable of antagonising the function of human MCP-1, demonstrating the high level of specificity of these aptamers and that the aptamers recognise MCP-1 in different ways. The aptamers may be used as a tool to further investigate the role of MCP-1 in inflammatory disorders and may also have a role as a therapeutic agent.

PMID: 11591377 [PubMed - indexed for MEDLINE]

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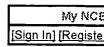
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Eotaxin-3 is a natural antagonist for CCR2 and exerts a repulsive effect on human monocytes.

Ogilvie P, Paoletti S, Clark-Lewis I, Uguccioni M.

Institute for Research in Biomedicine, Via Vela 6, 6500 Bellinzona, Switzerland.

Eotaxin-3 (CCL26) belongs to the group of CC chemokines that attract eosinophils, basophils, and Th2 lymphocytes. Like eotaxin (CCL11) and eotaxin-2 (CCL24), eotaxin-3 mediates its activity through CCR3. Here we show that eotaxin-3 also binds to CCR2 on monocytes and CCR2-transfected cells. In contrast to monocyte chemotactic protein 1 (MCP-1; CCL2), eotaxin-3 does not trigger intracellular calcium mobilization, enzyme release, or phosphorylation of the mitogen-activated protein (MAP) kinase ERK and induces a weak chemotaxis in monocytes. Instead, eotaxin-3 inhibits MCP-1mediated responses, thus acting as a natural antagonist for CCR2. This study also demonstrates that eotaxin-3 promotes active movement of monocytes away from a gradient of eotaxin-3 in vitro. This repellent effect is amplified when an additional gradient of MCP-1 is applied, demonstrating that the 2 mechanisms are synergistic. Eotaxin-3 effects on monocytes are largely abolished when cells are pretreated with MCP-1 or CCR2 antagonists. Like MCP-1-mediated migration, repulsion is sensitive to Bordetella pertussis toxin, indicating the involvement of Gi protein-coupled receptors. However, using transfected cells expressing CCR2 we could not detect F-actin formation or an active movement away induced by eotaxin-3, suggesting that either expression of a single receptor type is not sufficient to mediate cell repulsion or that the used transfected cell lines lack additional interaction molecules that are required for reverse migration. Eotaxin-3 was expressed by vascular endothelial cells and was essential for endothelial transmigration of eosinophils. Our data provide a mechanism by which 2 chemokine gradients that are oriented in opposite directions could cooperate in efficiently driving out monocytes from blood vessels into tissue.

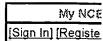
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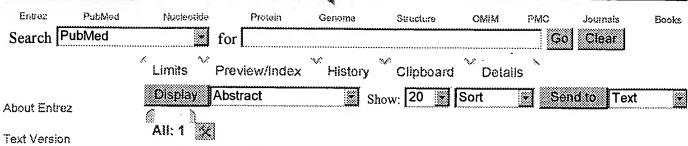
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CCR2: characterization of the antagonist binding site from a combined receptor modeling/mutagenesis approach.

Berkhout TA, Blaney FE, Bridges AM, Cooper DG, Forbes IT, Gribble AD, Groot PH, Hardy A, Ife RJ, Kaur R, Moores KE, Shillito H, Willetts J, Witherington J.

Department of Vascular Biology, GlaxoSmithKline, New Frontiers Science Park, Third Avenue, Harlow, Essex, UK CM19 5AD.

We describe here a classical molecular modeling exercise that was carried out to provide a basis for the design of novel antagonist ligands of the CCR2 receptor. Using a theoretical model of the CCR2 receptor, docking studies were carried out to define plausible binding modes for the various known antagonist ligands, including our own series of indole piperidine compounds. On the basis of these results, a number of site-directed mutations (SDM) were designed that were intended to verify the proposed docking models. From these it was clear that further refinements would be necessary in the model. This was aided by the publication of a crystal structure of bovine rhodopsin, and a new receptor model was built by homology to this structure. This latest model enabled us to define ligand-docking hypotheses that were in complete agreement with the results of the SDM experiments.

PMID: 12954060 [PubMed - indexed for MEDLINE]

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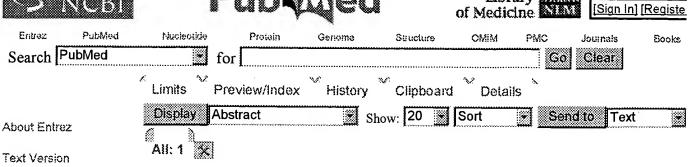
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Novel anti-inflammatory actions of amlodipine in a rat model of arteriosclerosis induced by long-term inhibition of nitric oxide synthesis.

Kataoka C, Egashira K, Ishibashi M, Inoue S, Ni W, Hiasa K, Kitamoto S, Usui M, Takeshita A.

Department of Cardiovascular Medicine, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.

Amlodipine (a new class of calcium channel antagonist) has been shown to limit the progression of arteriosclerosis and decrease the incidence of cardiovascular events. The mechanisms underlying the beneficial effects of amlodipine, however, remain unclear. Therefore, we hypothesized that amlodipine attenuates the development of arteriosclerosis through the inhibition of inflammation in vivo. Long-term inhibition of nitric oxide (NO) by administration of a NO synthase inhibitor, N(omega)-nitro-L-arginine methyl ester (L-NAME), to rats induces coronary vascular inflammation [monocyte infiltration, monocyte chemoattractant protein-1 (MCP-1) expression, increased activity of angiotensin-converting enzyme (ACE)], and arteriosclerosis. Here, we used the rat model to investigate the antiinflammatory effects of amlodipine in vivo. Treatment with amlodipine markedly inhibited the L-NAME-induced increase in vascular inflammation, oxidative stress, and local ACE and Rho activity and prevented arteriosclerosis. Interestingly, amlodipine prevented the L-NAME-induced increase in MCP-1 receptor CCR2 expression in circulating monocytes. Amlodipine markedly attenuated the high mortality rate at 8 wk of treatment. These data suggest that amlodipine attenuated arteriosclerosis through inhibiting inflammatory disorders in the rat model of long-term inhibition of NO synthesis. The anti-inflammatory effects of amlodipine seem to be mediated not only by the inhibition of local factors such as MCP-1 but also by the decrease in CCR2 in circulating monocytes. Inhibition of the MCP-1 to CCR2 pathway may represent novel anti-inflammatory actions of amlodipine beyond blood pressure lowering.

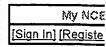
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Kataoka C, Egashira K, Ishibashi M, Inoue S, Ni W, Hiasa K, Kitamoto S, Usui M, Takeshita A.

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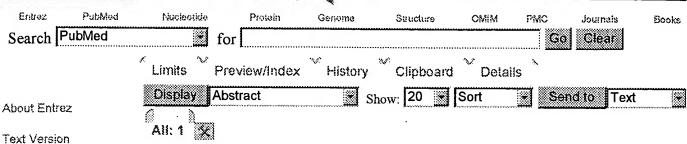
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High level expression, activation, and antagonism of CC chemokine receptors CCR2 and CCR3 in Chinese hamster ovary cells.

Parody TR, Stone MJ.

Department of Chemistry, Indiana University, Bloomington, IN 47405-0001. USA.

The specificity of leukocyte trafficking in inflammation is controlled by the interactions of chemokines with chemokine receptors. Reliable structurefunction studies of chemokine-receptor interactions would benefit from cell lines that express consistent high levels of chemokine receptors. We describe herein two new Chinese hamster ovary (CHO) cell lines in which the genes for chemokine receptors CCR2 and CCR3 have been incorporated into identical positions in the host genome. CCR2 is the primary receptor for the chemokine monocyte chemoattractant protein-1 (MCP-1) whereas CCR3 is the primary receptor for the chemokines eotaxin-1, eotaxin-2 and eotaxin-3. Both receptors are expressed at >5,000,000 copies per cell, substantially higher levels than in previous cell lines, and both are competent for binding and activation by the cognate chemokines for these receptors. Using these cell lines we confirm that eotaxin-1 and eotaxin-3 can act as an agonist and an antagonist, respectively, of CCR2. In addition, we show that eotaxin-2 is an antagonist of CCR2 and MCP-1 is an agonist of CCR3. Comparison of the chemokine sequences reveals several positions that are identical in MCP-1 and eotaxin-1 but different in eotaxin-2 and eotaxin-3, suggesting that these amino acids play a role in CCR2 activation.

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J Immunol. 2004 Apr 15;172(8):4977-86. PMID: 15067079 [PubMed - indexed for MEDLINE]

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Eotaxin-3/CCL26 is a natural antagonist for CC chemokine receptors 1 and 5. A human chemokine with a regulatory role.

J Biol Chem. 2004 May 28;279(22):23357-63. Epub 2004 Mar 23.

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CCL16 activates an angiogenic program in vascular endothelial cells.

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J Med Chem. 2003 Sep 11;46(19):4070-86.

PMID: 12954060 [PubMed - indexed for MEDLINE]

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J Immunol. 1997 Dec 1;159(11):5576-84.

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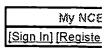
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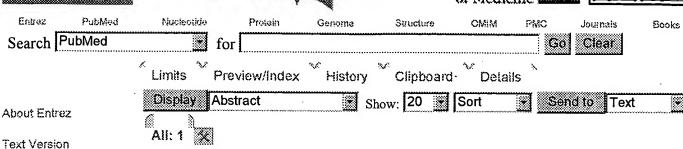
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Amino-terminally modified RANTES analogues demonstrate differential effects on RANTES receptors.

Proudfoot AE, Buser R, Borlat F, Alouani S, Soler D, Offord RE, Schroder JM, Power CA, Wells TN.

Serono Pharmaceutical Research Institute, 14 Chemin des Aulx, 1228 Planles-Ouates, Geneva, Switzerland. amanda.proudfoot@serono.com

Modification of the amino terminus of regulated on activated normal T-cell expressed (RANTES) has been shown to have a significant effect on biological activity and produces proteins with antagonist properties. Two amino-terminally modified RANTES proteins, Met-RANTES and aminooxypentane-RANTES (AOP-RANTES), exhibit differential inhibitory properties on both monocyte and eosinophil chemotaxis. We have investigated their binding properties as well as their ability to activate the RANTES receptors CCR1, CCR3, and CCR5 in cell lines overexpressing these receptors. We show that Met-RANTES has weak activity in eliciting a calcium response in Chinese hamster ovary cells expressing CCR1, CCR3, and CCR5, whereas AOP-RANTES has full agonist activity on CCR5 but is less effective on CCR3 and CCR1. Their ability to induce chemotaxis of the murine pre-B lymphoma cell line, L1.2, transfected with the same receptors, consolidates these results. Monocytes have detectable mRNA for CCR1. CCR2, CCR3, CCR4, and CCR5, and they respond to the ligands for these receptors in chemotaxis but not always in calcium mobilization. AOP-RANTES does not induce calcium mobilization in circulating monocytes but is able to do so as these cells acquire the macrophage phenotype, which coincides with a concomitant up-regulation of CCR5. We have also tested the ability of both modified proteins to induce chemotaxis of freshly isolated monocytes and eosinophils. Cells from most donors do not respond, but occasionally cells from a particular donor do respond, particularly to AOP-RANTES. We therefore hypothesize that the occasional activity of AOP-RANTES to induce leukocyte chemotaxis is due to donor to donor variation of receptor expression.

PMID: 10542293 [PubMed - indexed for MEDLINE]





J Immunol. 1997 Dec 1;159(11):5576-84.

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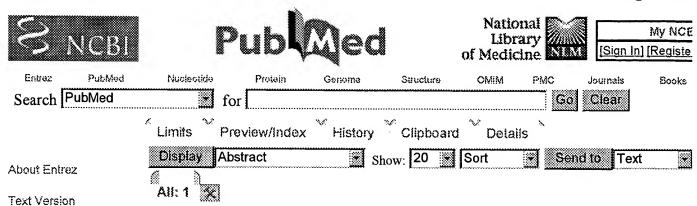


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Increased expression of the monocyte chemoattractant protein-1 in bronchial tissue from asthmatic subjects.

Sousa AR, Lane SJ, Nakhosteen JA, Yoshimura T, Lee TH, Poston RN.

Department of Experimental Pathology, U.M.D.S., Guy's Hospital, London, United Kingdom.

The expression of the monocyte chemoattractant protein (MCP-1), a member of the chemokine family of low molecular weight cytokines, was assessed by immunohistochemistry in bronchial biopsies from 12 asthmatic and 12 normal subjects. Both a monoclonal antibody (F9) and a polyclonal antibody were employed to detect MCP-1, while the mouse myeloma protein (MOPC21) was used as a negative control. Strong positive reactions for MCP-1 were seen in the bronchial epithelium. Subepithelial macrophages, blood vessels, and bronchial smooth muscle were also stained. Hue-saturation-intensity color image analysis was used to quantify reactions of the monoclonal antibody in the epithelial and subepithelial layers. With the monoclonal antibody, asthmatic biopsies showed 51.8 +/- 3.7% (mean +/- SEM) of the epithelium staining positively, whereas normal subjects reacted much less, with 6.4 +/-1.9% of the epithelium staining (P < 0.0001); there was no overlap between the two groups. Likewise, staining was increased in the subepithelium of asthmatic airway biopsies, with 11.5 +/- 3.1% and 2.0 +/- 1.0% staining positively in asthmatic and normal subepithelium, respectively, (P < 0.002). There was a significant correlation between staining of the epithelium and subepithelium (r = 0.77, P < 0.001). The polyclonal anti-MCP-1 antibody also gave strong reactions in the epithelium and subepithelium, with 34.0 +/- 7.8% of the asthmatic and 1.6 +/- 1.0% of the normal bronchial epithelium staining positively ( $P \le 0.0001$ ). These increased levels of MCP-1 in the asthmatic airways suggest that they may play a role in macrophage recruitment and activation and thereby contribute to the inflammatory pathology of bronchial asthma.

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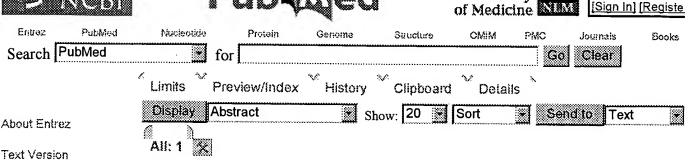
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Monocyte chemoattractant protein-1 in idiopathic pulmonary fibrosis and other interstitial lung diseases.

Iyonaga K, Takeya M, Saita N, Sakamoto O, Yoshimura T, Ando M, Takahashi K.

Second Department of Pathology, Kumamoto University School of Medicine, Japan.

Macrophages play a crucial role in the pathogenesis of idiopathic pulmonary fibrosis (IPF). To examine the mechanisms for increased monocyte/macrophage recruitment in IPF and nonIPF interstitial lung diseases (nonIPF) the localization of monocyte chemoattractant protein-1 (MCP-1) was investigated in 14 cases of IPF, seven cases of nonIPF, and seven normal control lungs (CTRL) by immunohistochemistry using a specific anti-MCP-1 monoclonal antibody, F9. By double immunohistochemical staining using F9 and one of the cell type specific antibodies significant differences in the staining pattern of MCP-1 were observed between IPF and nonIPF. In IPF MCP-1 was observed in cuboidal and flattened metaplastic epithelial cells, alveolar macrophages, and vascular endothelial cells. In contrast, no epithelial cells were stained for MCP-1 in nonIPF cases, although alveolar macrophages and vascular endothelial cells were labeled. Northern hybridization analysis of selected cases showed marked expression of MCP-1 messenger RNA (mRNA) in IPF and nonIPF compared with CTRL. These findings suggest that the MCP-1 production in IPF and nonIPF plays an important role in the recruitment of monocyte/macrophages. Monocyte chemoattractant protein-1 production by epithelial cells in IPF may be caused by the metaplastic nature of the epithelial cells and may be one of the key factors inducing the irreversible progression of IPF.

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Peptide mimics of monocyte chemoattractant protein-1 (MCP-1) with an antagonistic activity.

Kaji M, Ikari M, Hashiguchi S, Ito Y, Matsumoto R, Yoshimura T, Kuratsu Ji, Sugimura K.

Department of Bioengineering, Faculty of Engineering, Kagoshima University, Korimoto, Kagoshima 890-0065, Japan.

In this study, we attempted to analyze the peptide motifs recognized by 24822.111 and F9, monoclonal antibodies (mAbs) that inhibit the chemotactic activity of monocyte chemoattractant protein-1 (MCP-1), a member of the CC subfamily of chemokines. We isolated phage clones from a phage display library and identified six peptide motifs. One of these clones, C27, was strongly and specifically recognized by 24822.111 mAb, while another, G25, was similarly recognized by F9 mAb. Both the C27 motif and the G25 motif contain two cysteines in their sequences and have little homology to the primary amino acid sequence of MCP-1. These clones, however, bound to THP-1 cells, and the binding was competitively inhibited by MCP-1. The clones strongly inhibited the MCP-1-induced chemotaxis of human monocytes. The synthetic and intramolecularly disulfide-linked peptides of C27 and G25 (sC27 and sG25) also inhibited the chemotaxis induced by MCP-1, while their derivatives with serine in place of cysteine did not, suggesting the importance of the loop structure for the inhibition. These results suggest that sC27 and sG25 may mimic the MCP-1-binding domain to the MCP-1 receptor.

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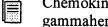


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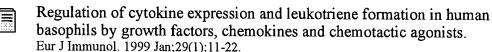
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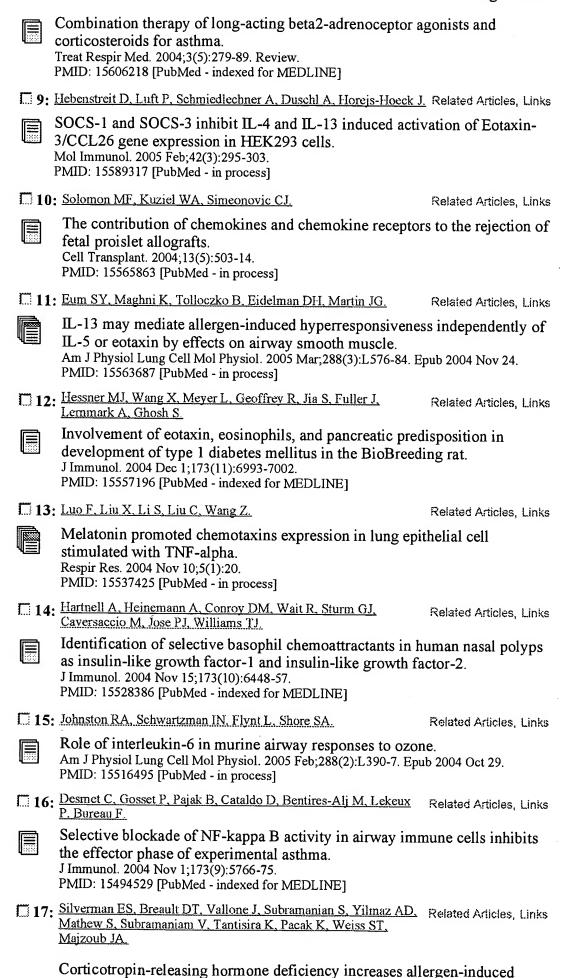
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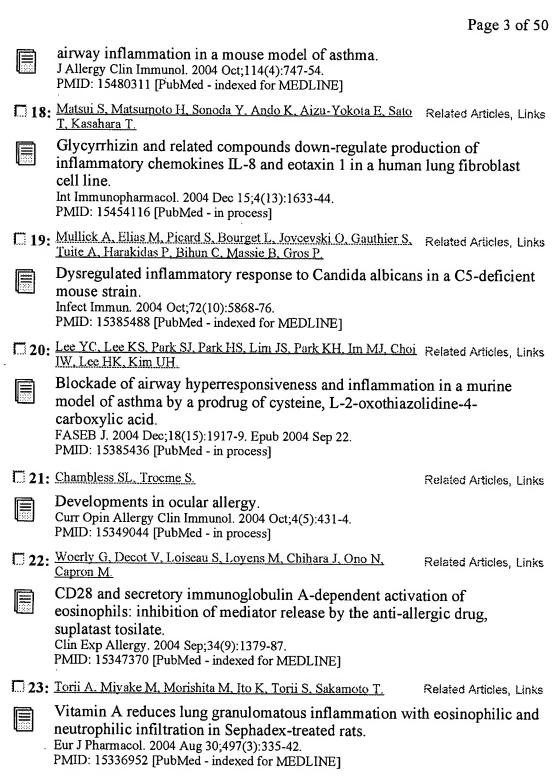
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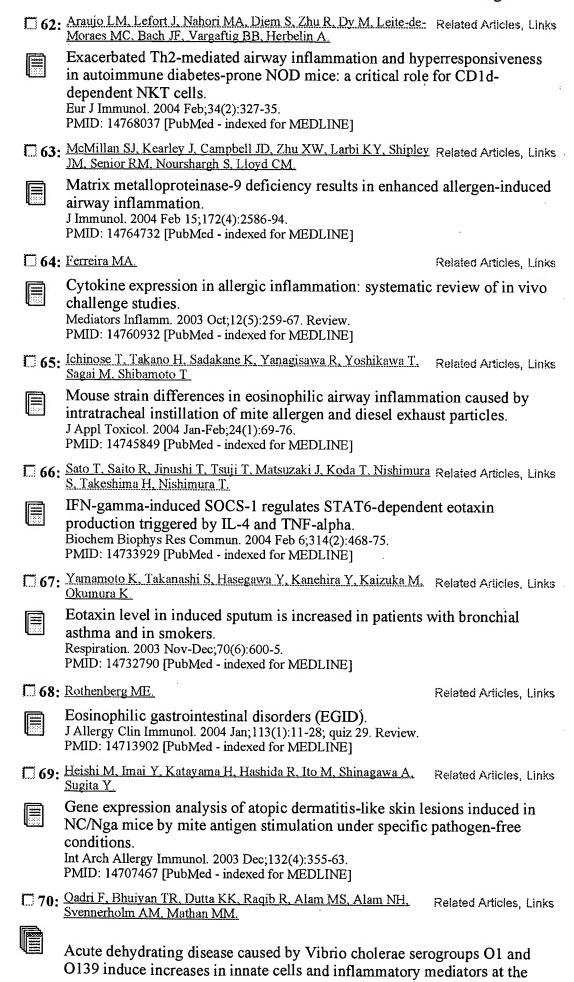
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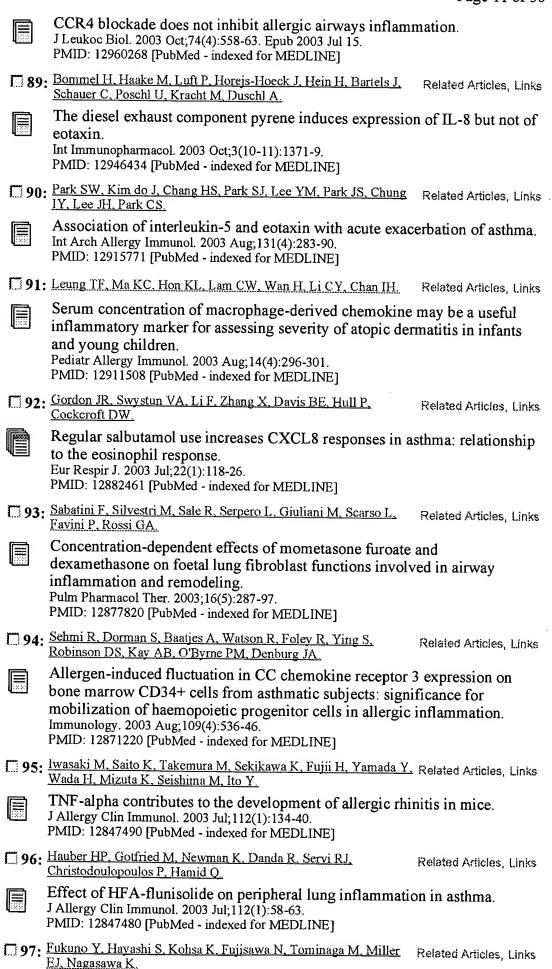
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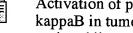


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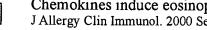
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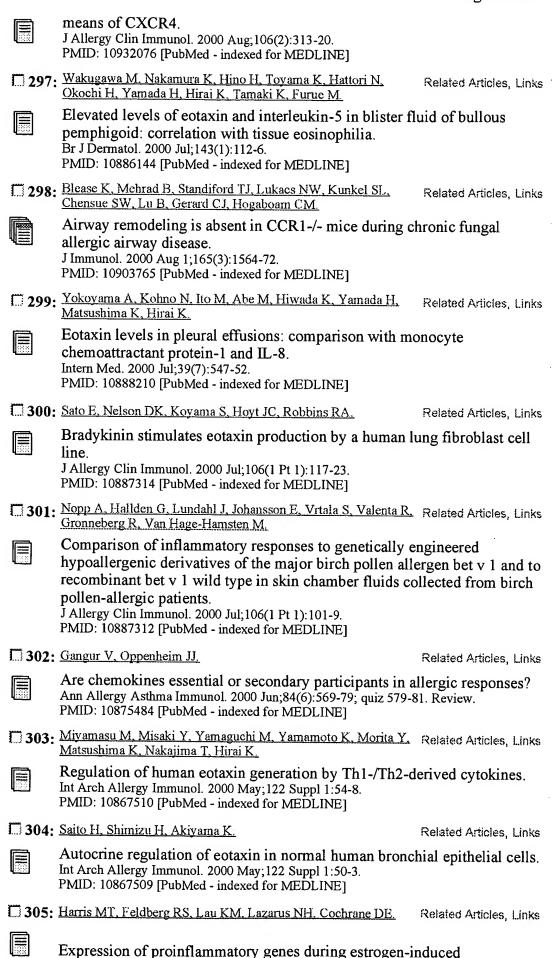
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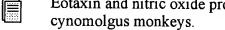
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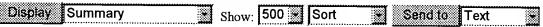
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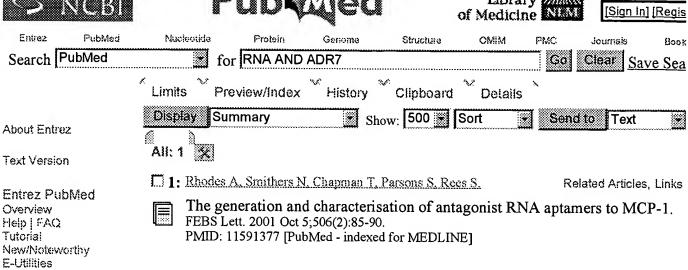
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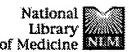
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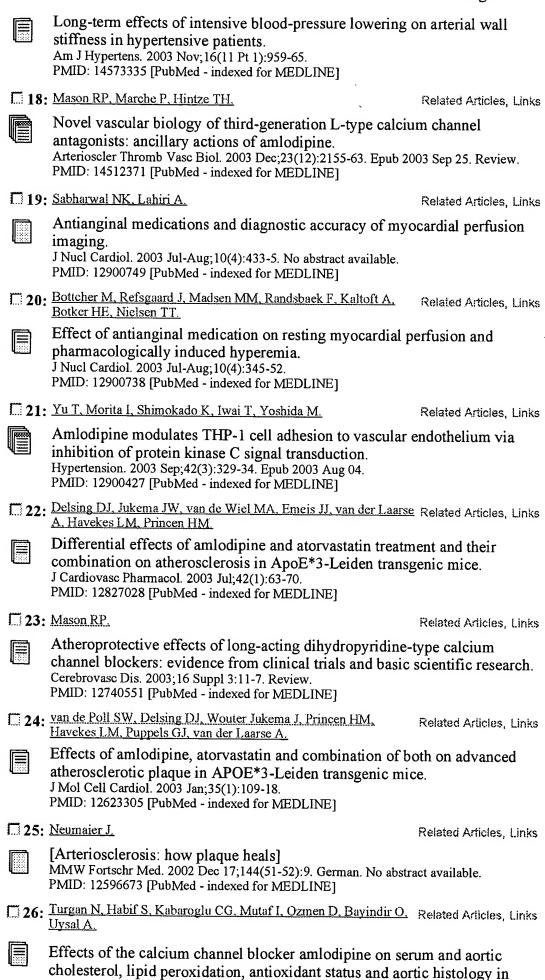
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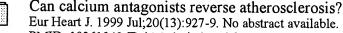


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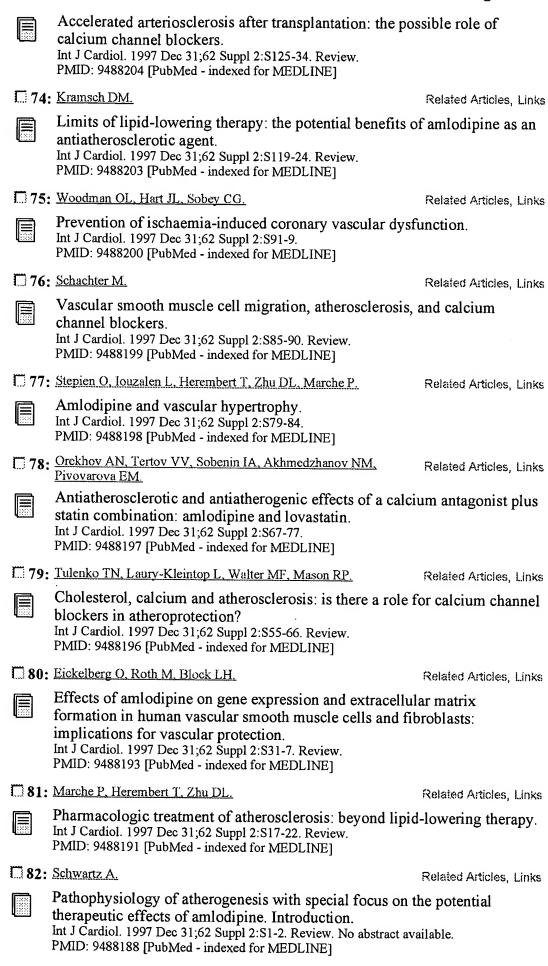
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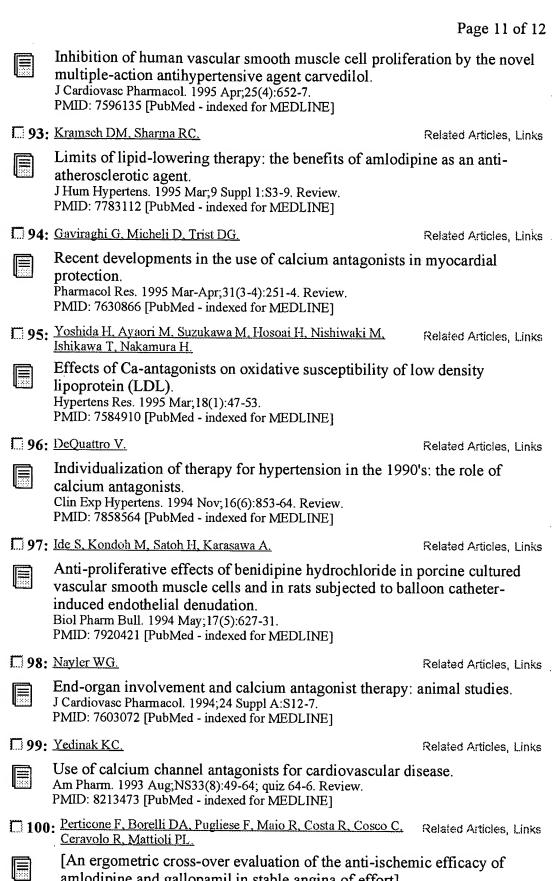
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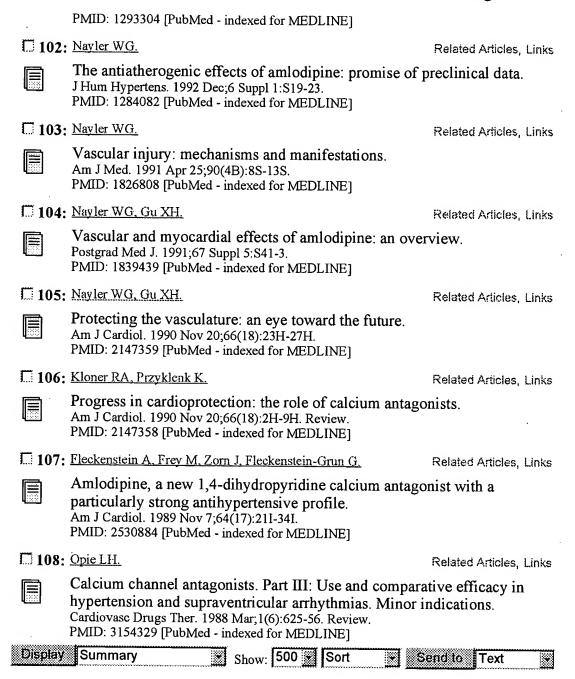
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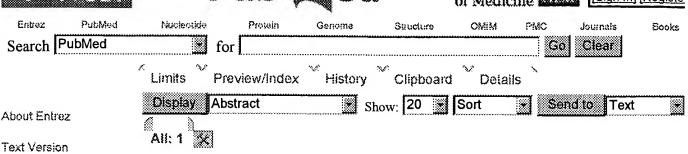
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The antiatherogenic effects of amlodipine: promise of preclinical data.

Nayler WG.

Department of Medicine, University of Melbourne, Austin Hospital, Heidelberg, Victoria, Australia.

Atherosclerosis is a complex and multifactorial disease, the endpoint of which is the formation of a calcified plaque. Intermediate events include intimal injury, smooth muscle cell proliferation and migration, macrophage infiltration, lipid accumulation and excess formation of ground substance. To determine whether the newly developed, long-acting calcium antagonist, amlodipine, slows the development of atherosclerotic lesions under experimental conditions, young New Zealand white rabbits were fed on a diet of 2% cholesterol plus 1% peanut oil for up to 12 weeks. Half the rabbits received 1 or 5 mg amlodipine/kg body weight/day. Amlodipine caused a significant and dose-dependent reduction in lesion formation in the thoracic aorta. At the same time thoracic aorta Ca2+ and cholesterol content were maintained at near normal levels, despite the raised plasma cholesterol levels. The protective effect of amlodipine persisted throughout a treatment period of 12 weeks, indicating the absence of tachyphylaxis.

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Antagonists: Antibody MCP-1 R02

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Propagermanium
Antibody 24822.111
Antibody F9
Eotaxin: eotaxin-2, eotaxin-3 but NOT Eotaxin-1
RNA ADR7
RNA ADR22
Eotaxin-3
Amlodipine

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Connecting via Winsock to STN
FILE 'HOME' ENTERED AT 17:02:14 ON 08 FEB 2005 => file BIOSCIENCE
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
FILE 'ADISCTI' ENTERED AT 17:02:31 ON 08 FEB 2005
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      Gong, Jiang-Hong; Clark-Lewis, I*
      Biomed. Res. Cent.,
CS
                           2222 Health Sci. Mall, Univ. British Columbia.
      Vancouver, BC V6T 1Z3, Canada
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     Monocyte chemoattractant protein-3, but not monocyte chemoattractant
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     Franci C; Wong L M; Van Damme J; Proost P; Charo I F
     Gladstone Institute of Cardiovascular Disease, San Francisco, CA 94110,
CS
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     HL52773 (NHLBI)
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SO
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     Journal code: 2985117R. ISSN: 0022-1767.
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     Signal transduction and ligand specificity of the human monocyte
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     chemoattractant protein-1 receptor in transfected embryonic kidney cells.
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     Myers S J; Wong L M; Charo I F
     Gladstone Institute of Cardiovascular Disease, San Francisco, California
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     94141-9100.
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Charo, Israel; Coughlin, Shaun
IN
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     Regents of the University of California, USA
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     CODEN: PIXXD2
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      Mammalian monocyte chemoattractant protein receptors
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      Charo, I. (Univ. California, Oakland, CA 94612-3550, USA)
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PCT Patent Appl. ( ***1995*** ) WO 9519436(Appl. US 08/182962 Filed 13
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      DNA encoding monocyte chemo-attractant protein-1 receptor - used partic.
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      Charo I; Coughlin S
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INCL INCLM: 514/356.000
INCLS: 514/355.000
NCL NCLM: 514/356.000
NCLS: 514/355.000
IC [6]

ICM: A61K031-44 EXF 514/355; 514/356

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